READ 39PG TRANSCRIPT OF 110min mp3-Mind & Matter - DNA & RNA Biology, mRNA Vaccines, Vax Contamination & Side Effects, Spike Protein, Ivermectin, Hop Latent Viroid _ Kevin McKernan _ #149

Welcome to the mind and matter podcast. I'm your host, Nick Jacobas. And today I'm speaking with Kevin McKernan. This is the 2nd time Kevin's been on the podcast. Kevin is the chief science officer and co founder at medicinal genomics.

He's been working in biotechnology and genomics for many years now. He took part in the original human genome project. He's been doing research in this area and helping develop technology in this area for a number of years at this point. His company does a lot of stuff to do with the cannabis genome and more recently the psilocybin mushroom genome. He's also done a lot of research and written quite a bit about MRN a vaccine technology and looked into some of the stuff to do with these mRNA vaccines that have been at the forefront of everyone's attention recently since COVID.

And we spent a lot of our time talking about that stuff. So we talked about some of the basics of DNA and RNA biology. We talked about what MRNA is and what it does naturally. We talked about how the RNA vaccine technology, these new vaccines that Pfizer and Moderna have used for COVID, how they work, how they utilize MRNA, how they differ from traditional vaccines. We talked about some of the work, some of the research Kevin and his collaborators have done looking at different aspects of these MRNA vaccines.

They've looked at the presence of possible DNA contaminants in the MRNA vaccine. So DNA sort of left over from the manufacturing process that is meant to be filtered out, but actually ends up being in some of these vaccines. We talked about exactly what they looked at, what they found there. He's done some research regarding whether or not the DNA left over in some of these vaccines can actually stay inside of cells in a petri dish, whether it can be replicated, whether it mutates, and even whether it might integrate into the host genome. And so we talked about, all of that stuff.

This is a controversial area. You know, mRNA vaccines, COVID, all that stuff. There's lots of noise out there about this stuff. It's pretty politicized area. If you're listening to the video version of this, I may not post it on certain video channels just due to the risk involved there. The video could get taken down due to the controversial nature of some of this stuff. But I asked Kevin about, you know, the science behind what he's done, how these things work, how

the mRNA vaccine technology is actually working, what he and others have uncovered about it it recently in terms of, what's inside of the vaccines, whether or not it's just the things that were designed to be in the vaccine products or whether there are any contaminants left over from the manufacturing process. We talked about the possibility of genomic integration, how this might relate to things like vaccine side effects and all of the stuff that's emerging about, you know, how this new technology is actually interfacing with our biology. We also talked a little bit at the end about hop latent viroid, which is an infectious agent that's causing a lot of problems for cannabis cultivators. And so we talked about what's going on there.

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The newest one is called sci Fox. It's an at home blood testing product. I get my blood work done every 3 3 months or so. I find that to be incredibly useful. I get a lot of really good biomarkers from that.

It's affordable. It's easy to use. It's way more convenient than going into the doctor, scheduling that checkup and and getting that blood work done. I'm a big proponent of, you know, taking your health into your own hands, preventative medicine, you know, knowing what's happening to your body as you change your your exercise, your diet, or your lifestyle. You can also become a paid subscriber on my Substack.

And with that, you know, I appreciate everyone's support, whether or not you're just tuning in or you're subscribing. And with that, here's my conversation with Kevin McKernan. Yeah. Why why were you why were you there? Was that, senator Ron Johnson having a hearing on, all this COVID stuff?

So you had, myself, Malone, Jessica Rose, Pierre Kory, long list of people who were, just presenting evidence on all of the shenanigans going on in COVID. Yeah. We're gonna talk about a lot of that, I guess here. Why don't we just start off with some basic stuff? Why don't you just tell everyone a little bit about yourself, your background, your expertise, and, you know, what you do at a high level?

Okay. Sure. So my background started in in this field in 1995, actually, on the Human Genome Project. I was started there as a member of the research and development team, and, shortly thereafter, 2 of the folks leading it left and left me in the reigns, wholly unqualified, and, I had to learn on the fly. So, I started managing that research and development group to about

probably a 10 or a 12 person group inside, the Whitehead Institute Center For Genome Research.

This is under, Eric Lander and Lauren Linton's guidance. And, we built basically the robotic platform and the DNA purification system to purify We did about 20,000,000 plasmids a year on that thing, to do Sanger sequencing for the Human Genome Project. So my role there was related to the automation and DNA purification chemistry and optimizing all the Sanger cycle sequencing stuff. And as that project came to a completion, a lot of companies were asking how to export that technology. And MIT held some patents on it, so we licensed those and spun them out to a company called Agincourt, which became a really large DNA sequencing company.

Actually it was the largest commercial sequencing entity I think in the world by probably 2,004. Beckman came to acquire it in 2,005. We had beyond just a DNA sequencing facility, it was a we had a bunch of DNA purification technology that was used to purify viruses and a variety of pathogens from blood. So they they acquired that. But in the process, there was a Skunk Works project we had to build a DNA sequencer that we used used to sequence DNA off of single magnetic beads.

And, that was starting to show some promise, but, of course, no one knew how to value it at the time. And so they decided to spin it out into its own company called Agencorp Personal Genomics. And a year later, we presented data sequencing E. Coli genomes at AGBT, which, caught the attention of ABI and Illumina, who proceeded to have a bidding war over the company. And ABI eventually, won that bid and purchased Agincourt Personal Genomics and brought the installed sequencer to market.

So I spent from 2,006 to about 2011 working at ABI, getting the solid sequencer to market. And, we also, towards the end of that, acquired Ion Torrent, which was another next generation sequencing system that worked on semiconductors. So I worked on that program, helping get that developed and and out the door, and then decided to, you know, split ways and and work on some other passions of mine, one of which was the cannabis genome. I had been I had a very large non compete the company was now part of Lifetech, which was a entity, which meant I couldn't really compete. My noncompete meant I really couldn't work in that space, so I kinda just went off into the ag space.

And, we started sequencing, cannabis genomes because we felt they were it's a genome that hadn't been sequenced at the time, and, it had all these therapeutic compounds in there that could be helpful for cancer. And, so we figured let's get the thing public and see if that helps kinda mature that field. That field got a little complicated, as you might imagine. Growing a

business in the cannabis field is difficult from a banking perspective, and and the laws keep changing on you. So the company kind of pivoted back into doing clinical sequencing of of people who might benefit from, cannabinoids.

So a lot of, epilepsy patients, mitochondrial disease patients, autism spectrum disorder. So we were doing exome sequencing on those, cohorts as a clinical test. And that went on for about 5 or 6 years before we realized that cannabis market started to devolve and mature, and we pivoted back into doing, cannabis testing. So right now, my role at medicinal genomics is building PCR tests that target all the pathogens in cannabis that can either destroy it from a yield standpoint or impact patient health. A variety of jurisdictions around the world demand E.

Coli, salmonella, aspergillus. A host of pathogens get tested for every, every pound of cannabis that's sold. And we don't do the testing ourselves. We just make the picks and shovels that other labs use, to do this type of testing. So we're mostly involved in assay development and genomic sequencing still to this day.

We we do a lot of, genomic sequencing of cannabis genomes, and it's about 2,000 of those public now on our website. And, we've extended that to other medicinal organisms. Psilocybe comes to mind because it's has a similar, I'd say, therapeutic profile for although it works in very different ways, it's one of these medicinal organisms that is, I'd say, understudied in the current, FDA regime, if you will. Hard to get hard to get patents on these natural products, so a lot of people push them aside. And, so we started sequencing.

We've we've been through about a 100 of those genomes that are up on our website, and publishing papers on that topic, fairly recently. So, yeah, I don't belong in sequencing vaccines. I can fish out of water, I suppose. But, it, it came to us somewhat serendipitously. And, I think because maybe your podcast and a few others highlighted some of our work in evaluating some of the early COVID tests, we were shocked that these COVID tests came to market without internal controls, because we could never get away with that in any other market. And so, without those internal controls, it's really hard to gauge your viral load, and it seemed odd that we were racing these tests out the door without that. And perhaps there was a higher positivity rate early on in the pandemic. Many of the companies cleaned that up over time, but the kind of horse was out of the barn at that point. And there's So I wrote a paper on that and one with Peter McCullough, which I think caught your attention on the last podcast just talking about some of the differences between these vaccine MRNAs and, and and what was actually in the virus, which led to some discussions about frame shifting, which I think have recently been shown to be correct. There's a paper from Mulroney that came out

showing that the pseudo uridine can cause some frame shifting that is probably not, the same type of frame shifting that you would see in the virus because the virus doesn't have those types of slippery bases in it.
Yeah. So Yeah. I wanna get to that. Let's spend just a little time giving people who don't have the background that you have, some vocabulary and just some basics that they can keep in mind. Let's start super basic, but I don't want to spend too much time here.
DNA versus RNA. So there's DNA in all of our cells. There's RNA in all of our cells. High level, what's the difference between DNA and RNA in terms of what they do and what they're made out of? Yes.
I think a good analogy is DNA is like what's on your hard drive on your computer, and RNA is like what's in your task manager, like what programs are actively being run from your hard drive. And so they're often fragmented and smaller in size, and they're ephemeral. They can go on and go off. So your DNA is this hard drive of all the programs your cell can run. And then the RNA, whenever it wants to run a program, it has to turn that program into RNA for the cell to then turn it into proteins.
So you have to think of it as all the programs possible that a cell can run, and not all cells are going to run all programs at the same time. That's in fact what makes the cells very different is they selectively choose certain programs to run-in order to be a heart cell versus a liver cell. Cell lines, which you can measure by sequencing the RNA. So the RNA will tell you what genes are actually turned on in a given cell, and how loud are they being turned on? Do we have one copy of the RNA?
Do we have a 100,000 copies of the RNA? Now the RNA is supposed to be fairly ephemeral. There's all types of circuitry in the cell to express it and then destroy it so that you don't have something that's constituent turned on all the time. But that process is quite delicate and, is the process that, some of these vaccines are trying to hijack to get spike protein made. Yeah.
Okay. So so DNA turns into RNA. The RNA can be likened to the programs your computer might be running at any given time for a little while, for longer periods of time, they shut on, they shut off. The RNA, the mRNA is made from the DNA. The mRNA can then be used to make proteins from it.
Yes. In terms of the code here, we we talk about the letters of the DNA and the RNA code. What are those letters, and how do they differ between DNA and RNA? Well, A, T, C, and G are the ones that are known to be in DNA. There are some exceptions.
You sometimes have methylated versions of these bases, and you sometimes have uracil, but generally uracil is mostly found in RNA. So when certain bases in the DNA get damaged, they

might appear as a uracil. But, there's a whole pathway meant to clean that up, to get rid of these, thiamidines that have been turned into uracil. But RNA is always replacing that t with a u. So whenever you see RNA sequence, it would be a u c g.
There tends to be a U replacing the T. So that's one key difference in the language. You oftentimes find other modifications in RNA as well. There's a very rare type of alteration to the U, who's known as pseudouridine. About 0.6 to 1% of the of the Us in any given RNA have a pseudo uridine in them, and the cell can further methylate that into N1 methylsudouridine with a set of enzymes that methylate the pseudouridine.
So there's a pathway for about very small infrequent percentage of the RNAs have these pseudouridines which are you know, we don't fully understand their role in biology, to be honest. When you do knock out mice, when you knock out the enzymes that that play in this cascade, really weird things happen. But I think it's important for folks to know that it's mostly relegated to snoRNAs and tRNAs. It's very rarely found in messenger RNAs, these pseudo U's, but they do exist in nature. And both of the mRNA platforms leveraged the fact that that base was very rare and different and replaced all of the Us in their mRNA with this N1 methylpseudo U for reasons of wanting to keep these RNAs around longer.
The enzymes that we have that that turn these RNAs on and and then destroy them are a little bit slower to act on the pseudo the N1 methylpseudouridine in the in the Pfizer Moderna vaccines. That was considered a feature, not a bug, at the rollout of this because their largest concern was injecting these RNAs and then having your cells destroy them before they could express spike protein. So so mRNA is A, U, G, and C. Most of our RNA, most of the time uses uracel as the u, but there's a slightly different version that can be incorporated. And that happens naturally a small percentage of the time.
Yes. And that affects probably among other things, how long those mRNAs are lasting in the cell. Yes. There's some literature suggesting it may play a role in cellular as well. The ones that are have pseudo uridine on them tend to be more nuclear localized, but, that's not that's not a hard and fast rule.
And so in terms of the the mRNA vaccines, the Pfizer and Moderna vaccines that we're pretty much all familiar with at this point that have been used for COVID, At a high level, what was the intention behind these vaccines? How were they meant to work in contrast to traditional vaccines? Jason Brett (zero twenty:fifty four): So traditional vaccines would put a protein, in your body, be injected into your arm, and your body would build immune defenses against that that circulating protein. And it's not meant to circulate. It just be in your arm such that your immune system could see it and build antibodies.

The approach here was instead of having to use a protein is was it was to inject an RNA and have your cell make the protein for them, if you will. Now that invites a lot more variation, if you will, or variability because not everyone translates RNA at the same speed. We don't know if they're all gonna get when the protein gets made, if it's gonna fold the same in every person, if it's gonna get presented on the cell the same way. So you're several steps downstream in the manufacturing process that you're sort of outsourcing to the patient and having their cells build these RNAs. Now I think one key difference here is that when your cells are making these proteins, they're somewhat painting a target on their back for your t cells to come and destroy them.

So in the case of a traditional vaccine, you're not decorating your cells with the antigen to have your immune system attack your own cells. You're just teaching your immune system how to defend against this antigen, but you're not really decorating your own cells with it. So, I think one of the risks that we're seeing with these mRNAs is that when your cell when your own cells express these foreign proteins, they become targets for destruction. And that seems to be what may be happening in myocarditis. We have this cross in paper out that shows there's mRNA and spike protein in heart tissue, and there's a lot of inflammation in that heart tissue.

And they can detect it 30 days later. So it may be the immune system's getting turned on against any cell in your body that's expressing these things. And, you know, that could be if those are the wrong sets of cells, that could be very damaging. One of the other issues with these is that there's some biodistribution studies that show the LNPs aren't really contained to the arm. They can go all over the body, so you don't really know which organ you're painting with these things.

If you happen to this happens again to your circulatory system, it could paint the epithelium of your circulatory system, and then that epithelium gets destroyed. That could be causing clots. Yeah. Mark Girdo did some interesting work on this. His bolus theory, I think, has some has some legs to it, that you're stripping the epithelium, of your, your circulatory system, and that's leading to all types of, leakage, if you will.

That can happen on on the blood brain barrier. It could happen in your, in your aorta. It could happen in a lot of places that you don't want it to to occur at. So I think there's a difference a key difference is the protein is traditionally, the proteins injected probably does a better job staying localized. It doesn't express it's not made inside your cells and thus painting targets on your own cells, thus leading to destruction of your own cells.

I think those are 2 very key differences. And the third one is that, by asking human cells to make the protein, we're now exposed to all the variability in the human genome amongst the population. Some people may make those proteins more effectively than others, and there may be much wider variety in the expression levels of the actual antigen than just giving someone a really well known concentrated dose a of a peptide. So so with a traditional vaccine, you're injecting a protein or a set of proteins from a pathogen directly into the body. You know exactly what the dose, how much of each of those proteins is in there.
And it's just those literal proteins. You're not, you're not, asking your own cells to produce that protein. The proteins are getting into the body directly. And then an immune response comes to that. And, and, you know, that trains our immune system, so that when we encounter the pathogen in the future, if we do, it's preempting that immune response.
And that's how vaccines work. The mRNA vaccines you're saying is we're injecting mRNA instead of the protein. The mRNA is by design meant to go into our own cells. Our own cells machinery is then producing protein from that. In this case, that would be the SARS CoV-two spike protein.
But what you're saying is that now the protein is being made within, and as you said, painting our own cells. And one of the other things you said is there's probably going to be natural variability in how much protein you produce or which proteins you produce. Perhaps you're making the spike protein and some other variants just based on differences in the biology, from person to person in terms of how quickly the translation is happening, how exactly that protein is folding and that type of thing. Hypothetical concerns because we're asking the cells to do the manufacturing for us, but, the pharmaceutical companies didn't do a very good job proving that the cells made those proteins faithfully. They just showed we have antibody response, which, there's we now know there's there is high variability in this due to this base that they put in.
So they put in this N1 methylsutouridine so that it would evade the immune system and then last longer. But that came with some compromise, which is that the ribosomes that read RNAs that have that many modifications, get confused, and they sometimes slip and get out of frame, and make I think the Moroney paper said 8% of the proteins were frame shifted. So we're already taking an 8% loss in fidelity making unknown proteins, to get the spike manufactured by ourselves. Wouldn't be the case if you injected a purified protein. You'd be able to make it outside of the body, purify it, quantify it, and put in only what you're looking for.

But when you start asking for your cells to perform its manufacturing for you and you have to, you know, put some camouflage on the RNA to sneak it through the immune system, you're inviting, some some fidelity issues with the translation process that has now been exposed through, the Moroney paper. And so just to tie some of this stuff together for people, mRNAs naturally are, as you said, they're ephemeral molecules. They're not supposed to last very long in the body. They're supposed to be produced for set periods of time. You don't want them sticking around too long.

So our bodies have lots of enzymes to quickly break them down. It sounds like what you're saying in terms of the mRNA vaccines that were manufactured for COVID, they used this pseudo uridine in place of the uracel. So they they use that slightly different version of the u in the RNA code. And that's because, my understanding is if you use the normal u, that the mRNA is not going to last really long enough. It's going to get broken down right away, and you won't actually generate an immune response.

So it has to do with increasing the stability of the mRNA, so it actually sticks around a little bit longer. Yeah. So there's a co class of RNases, that localize in different cell compartments and tissues. But the one that Carico was really concerned about was something known as RNase L, and they demonstrated that RNase L was less active on mRNA that had this modification to it. So they thought this is a great way to get the RNA to last longer, and they were right about that.

It does last longer. But I think maybe the concern that the models didn't predict is that clearly in some patients it's lasting a lot longer than even They were suggesting 48 hours. We've now had papers out showing it, 28 days in plasma, 30 days in the heart, 5 days in breast milk, 10 days in placenta. So they're picking up this mRNA, you know, anywhere between 5 to 30 days later in various tissues that have been surveyed. The spike protein itself is sticking around longer, and, there's one paper out showing 187 days where they're picking up spike protein. So I don't know if that's the RNA is still around and we're not detecting it and it's still expressing or if the spike protein is really hard to degrade. I think there's still a lot questions to be answered as to what's the mechanism of action of that persistence. But, it's possible that these LNPs are getting to stem cells, which are immune privileged. So your immune system won't attack your stem cells. And if you happen to get an LNP into a stem cell, well then you kind of have it camouflaged inside your body, and it could be expressing spike protein for much longer than, predicted.

What is an LNP? Oh, sorry. Lipid nanoparticles are the it's kind of the fat bubble they put these RNAs into so they can get into your cells. That means it's protected from a lot of the nucleases

that might degrade it outside of the cell, and it it kind of Trojan horses its way right into a cell. Okay.

So so a lot of a lot of the design of these vaccines was aimed at making sure that the mRNA actually got into the body and lasted long enough to do what we wanted it to do. Yes. Yeah. That's it's key. And I don't know if much attention was put into understanding the clearance of it.

You know, how Having something last for a long time may not be a desired outcome. Most immune responses are finding a small antigen and preparing the body to amplify its response to that the second time it sees it. So you don't necessarily need a lot of antigen to deliver, to get to get to build a response. I think we were in such a new space here. Their concern was, let's make sure we at least get a response.

So let's make the mRNAs last long without as much concern over what happens if they last too long. And, this persistence create disease. It may maybe it wouldn't create disease if it wasn't expressing a protein as notorious as a spike protein. Maybe it's maybe the platform's fine if you had some other type of, you know, benign protein in there. But the combination that we that there's a protein that now has a lot of publications on its toxicity, and persistence is one concern.

Now there's other concerns out there that, well, what if you just had naked L1Ps with nothing in them? What damage would that do? We don't have an answer to that. It could be that just these L1Ps bombarding these cells with any foreign peptide turns the immune system against them, and you're really just inviting the immune system to erase a certain percentage of the cells. I mean, there's some numbers on how many LNPs are in this.

I've seen literature that it's anywhere between like 50,000,000,000 to a trillion. I tend to think the 50,000,000,000 number is more accurate based on just surface area volume calculations I've done, but that's still, you know, 50,000,000,000,000, you probably have you know, 40 trillion cells. So you're talking about 1 in a 1000 cells getting painted for destruction, which if it's the wrong cells, that can be a problem. So when we talk about the the mRNA vaccines for COVID, they contain the mRNA, which encodes the spike protein. But it's not the, it's not identical to the native mRNA that's in the virus.

It's using this modified U in the code. And they're also encapsulated in these lipid nanoparticles. So it's not just like we took the straight mRNA chunk of mRNA from the virus that encodes the spike protein. We took that, we modified it and tweaked it. We wrapped it in these protective lipid nanoparticles, and that's what goes into the body.

Yeah. And that that actually is an important point because, as I'm sure we'll get into, if you start having contaminants that are in these LNPs, some of your defense mechanisms to get rid of them can't do their job. So when you wrap this in a fat bubble like that, it protects it from a lot of the nucleases in the blood. That's true for the RNA and any potential contaminating DNA that's in the shots. Typically, if you inject DNA into somebody, it has like a 10 minute half life in the blood.

It's not a big deal. They have a lot of previous vaccines that have had DNA contamination in them when they're injecting those peptides, but that stuff is, gets destroyed pretty quickly. The moment you package it into an LNP, you're bypassing that whole defense mechanism, and you're delivering that DNA and that RNA straight to a cell. So now it's there's an unknown as to whether you know, what the tolerability is, how much DNA can we tolerate under those circumstances. That that hasn't really been addressed by the FDA.

In basic terms, can you walk us through just the basic process by which Pfizer and Moderna manufacture the mRNA vaccines? How do they go from raw materials to the final product?

Well, it starts with your initial question. So they start with DNA, and they take an RNA polymerase to express RNA off of that DNA. And so they use that DNA almost like a it's only called a template, but it's almost like a printing press.

You have a system that you can just print RNA off of. Now, there's 2 different ways you can generate that DNA. And Moderna from the beginning had a plasmid that was making their RNA. Their trial went on with the plasmid. Their clinical trial reflected their mass production. Pfizer made a bit of a switch here. They started not having enough DNA, so they PCR amplified the region of DNA that they wanted the RNA to make out of a plasmid. So usually what a plasmid is is a circular piece of DNA that allows that DNA to replicate and stores very well because it's circular. You can put them in bacteria and bacteria can harbor these plasmids. And you just grow the bacteria out and it creates about a 100 of these plasmids per cell, and the E. Coli cells double every 30 minutes if you give them the right temperature and nutrients. So it's a great system that's been used for ages in the biotech system to replicate DNA inside of another organism. So Pfizer ran their clinical trial PCR amplifying off that plasma DNA, and then they made RNA from the PCR product. Now the reason that's materially different is that when you amplify a plasmid like that, you can then your amplified material is about a 1000000 times higher in concentration than your background. You could put a very, very small amount of plasmid in, amplify it, and get a 1,000,000 fold amplification in about 20 cycles PCR.

And that means that your contamination is a millionfold diluted. So then you can then take that very clean DNA and make RNA from it. And now when you're done making that RNA, you

now have a pot that has DNA, some DNA template, and lots of RNA that you just made. And conventionally, they would like to erase that DNA, and they use some enzymes like nucleases to get rid of that DNA. And that is something that seems to be failing in their process.
There seems to be a failure to get universal, DNA, since the enzyme they use, so to completely eradicate this DNA. So in the process of scaling this up, they they went they did the trial on this PCR generated material, which is very clean. And then when they had to scale up, they switched the process to process 2, which skipped that amplification step. And they tried to get the plasma DNA directly into the into the RNA generation process. And since they skipped that step, what that means is the complexity of the background DNA is now the entire plasma, not just the region you amplify.
So this means another, like, 4,000 bases of DNA come through that have an antibiotic resistance gene, that have an SV40 promoter, that have they have a variety of other components in the backbone of this plasmid. So there's more background genetic material that comes through when you do this, when you skip that PCR step. Now they were supposed to do a study comparing process 1 to process 2 across 252 people, and they threw the towel in saying it's not going to matter. It's not big enough of a study to really find anything. And the EMA looks like they let them off the hook on that.
So we don't really know, if there is a different adverse response rate, that would be witnessed in process 2 versus process 1. That that's been something that, Retzit Levy and Josh Gutzkow have have brought up in the BMJ showing that this is, this is unusual in the biotech space. And with complex biologicals like this, the actual process is the product because there are so many different components in living systems like this that when you're using E. Coli to amplify your DNA, you can have a host of different contaminants that you can't necessarily measure that can come through the process. So whenever there's a process change, they consider that to be a new product because you can't fully characterize everything that might be in that background.
Now, one background in particular a lot of people highlight is when you're working with plasma DNA and you don't amplify it, you have to crack open those E. Coli cells and get your DNA out of those cells before you make RNA from it. And that process can be prone to leaving a lot of E. Coli guts in the equation. And by guts, most people are concerned about endotoxin that comes through on the coat of the E.
Coli cells. Crack open those cells, and now endotoxin, which is known to be a really aggressive immune stimulator, comes through with the plasma DNA. That can be very tricky to measure, nature of that compound. But that's something that is if it is there, we don't know how much

of it's there because most of the documents we're finding have the endotoxin levels redacted. But if it's there, that is known to create anaphylaxis anaphylactic shock.
So that is something that could be responsible for some of the acute reactions that people see. Mhmm. I don't think the DNA contamination is creating any of these people fainting or anything acute. It's something that might be more long term of a concern. So so when we think about so if someone goes in and get gets their vaccine, the mRNA vaccine, you know, I want to talk about what's in that syringe.
So there's the stuff that's supposed to be in there by design, and then there's the potential that other things that we don't want to be in there that aren't supposed to be in there are also in there. Starting with the first group, the stuff that's in there by design. You've got obviously the mRNA, which has this modified u, in the code that we discussed. You've got the lipid nanoparticles, the little fat bubbles that are like little protective shells around that mRNA. Give us a sense in a single dose of, say, the Pfizer vaccine, how many mRNA molecules are in there? How full is it with these lipid nanoparticles? And what else is in there by design? So there should be about 13,000,000,000,000 13 to 14,000,000,000,000 mRNAs in a Pfizer dose. Moderna's got 3 times that amount. They're closer to 42 trillion, mRNAs.
And those are we estimate are probably in, you know, 40 to 50000000000 lipid nanoparticles. So, yeah, do the math on that. It's a couple 100 to 1,000 of these mRNAs per, per LNP. There's some cholesterol in PEG and other ingredients that help stabilize these LNPs, but I think those are the 2 key things is that the the mRNA is there and the lipid nanoparticles there. What we discovered is that there's also DNA inside those LNPs, and that's broken up, it's fragmented, but it's, at there's billions of copies of those as well.
Not trillions, but billions. Yeah. So so you've got, billions with a b of lipid nanoparticles in, say, a Pfizer vaccine dose. Each one of those is gonna contain on the order of 100 of mRNA molecules. So you got 1,000,000,000 of little fat bubbles, trillions, it sounds like, of mRNA molecules.
How did you guys go about looking for DNA contaminants that were in there? What was that process? How did it start? Yeah. So we were we were actually studying HOP latent viral infections in cannabis.
This is something that's devastating the cannabis field and we're doing just boatloads of RNA sequencing of plants that were infected at different points in the infection cycle. And when you do RNA sequencing, as we mentioned before, you should get sequencing that lines up only over the genes. And if you get sequences that aren't in the genes, there's probably something wrong with your RNA sequencing system. And and that and one one week we

came in, and that's what happened. We saw sequencing that was all over the genome, and we're like, okay.
Something's broken. We're not capturing mRNA. We must be capturing genomic DNA or or somewhere there's a problem. We shouldn't be getting sequencing all over the genome like this. So to solve that problem, what you typically do is you spike in a known mRNA as a control.
If you can't capture that, then you can pinpoint, Okay, the magnetic beads that pull down the RNA are broken or maybe the DNA step is broken. So I needed an mRNA had that was pharmaceutical grade, had a poly A tail. And instead of ordering 1, I was like, Well, I've got one of these on the shelf. Someone shipped me. That's a Pfizer vaccine.
That should be pharmaceutically pure. Let's pop that thing in there. And if that doesn't come through our RNA sequencing pipeline, then we can figure out what's broken about it. It did come through the sequencing process. We did identify we had a bad DNA enzyme that wasn't chewing up the background DNA, which is why we were getting sequencing everywhere.
But in the process, it also revealed that there was the Pfizer's vaccine plasmid was still in the vials. So we ended up with So the piece of circular DNA that they use to amplify to get the mRNA, a piece of that was still in there? That was still in there. Billions of copies of it per vial, or per dose, I should say. So that was a bit shocking because we weren't expecting to find any of that.
We got these assemblies back that had spike protein in there, and we're like, Well, spike should be 4200 bases. Why the heck is this thing 7800 bases long? And we threw it into SnapGene, and that's when we saw, oh, there's an s p 40 promoter. There's a Kenamycin gene. Then this is their expression factor.
You saw you saw the you saw the, the carcass of the DNA plasmid and the things that we know are in it. Yeah. Their top secret, blueprint of how to make it, basically. Oops. No.
So so how how many of these experiments did you do? How how fresh was that vaccine batch that you had? Is how confident are you basically that Oh, yeah. So that's a that's a great point. That's something that people always bring up.
So they weren't very fresh. Actually, people shipped these to us, and I ignored their request to sequence them for probably 6 months, chucked them in the freezer, and forgot about them. And then when I had an emergency, I was like, Oh, that thing will work. And by the time I pulled this stuff out and used it, it was in fact an expired vial. Now people have since gone back and replicated this with non expired vials.

Philip Buchholz did some of this work. David Speaker did this work in Canada as well. I see. So so other people independently made this observation using different fresh batches. Which has been very helpful because people had good reason to throw tomatoes at us, if you will. When we published this, they were mad that we had an expired vial that we that we sequenced. But, you know, the these, there's no reason to believe the expiration date would actually destroy the DNA or make more DNA in there. Like Right. Right. Yeah.
It's 20. You wouldn't expect an old batch to have a DNA contaminant that wasn't in the fresh batch. Right. Right. Unless someone some gremlin got in there and put it in there.
But the fact that it was Pfizer's expression vector was a pretty good fingerprint that Pfizer put it in there. It had their spike sequence in it, and it had, what looked like a valid expression vector. And the other thing to know is that the expiration dates weren't some hard science. They often would just change them and announce that, oh, this expired vial can now be used. So expired vials were injected into people.
That didn't stop them from using them in the field. It just, was something that was a critique of, the, you know, the way we went about sequencing this. And that's just because we didn't we didn't set out to to sequence this as any type of grand experimental plan. It was kind of an accident. And so what we did to try and help there is we we built PCR assays to make it really easy for other people to replicate this in other places, and so they wouldn't have to go through this expensive sequencing process.
And that, that indeed helped, the replication of the work in other places. So the implication here is that in the mRNA vaccines that were actually used, you had not only the mRNA and the lipid nanoparticles, all the stuff there by design, but you also had remnants of the DNA from the plasmids used in the manufacturing process of these vaccines. I guess the next question is, how big of a concern is that? Is it plausible that those are going to cause an issue in a human being? Or are these DNA sequences likely to be pretty inert and not really doing much of concern?
Jay Gould (4seven zero six): So I think that's where a lot of the debate lies is what's the clinical implications? I don't think anyone's doubting their existence anymore now that there's been so much replication. We've had the EMA, the FDA, and Health Canada come out and admit that, Okay. Yeah. This could be in there.
They trust the manufacturer who's measuring this to say it's below some certain limit. I mean, Philip Buchholz brought up a very good point on this, which is your limits were set based on the decay rate of natural DNA being injected in traditional vaccines. This is a different beast.

We have them in LNP, so they're not going to decay. And the transfection efficiencies of this DNA is going to be very high.
So the prior regulations on this around 10 nanograms of DNA per dose. Now, those guidelines have changed a thousand fold over the last couple decades. In the Reagan era, they put in the NCVIA Act, which is the National Vaccine Injury Act. And that gave pharmaceutical companies a bit of liability shield on vaccines. So since then, the regulations have moved from 10 picograms up to 10 nanograms with traditional vaccines that don't have LNPs.
So we're in a different world now where we have LNPs that are facilitating the DNAs entering the cell, and arguably, that limit should be revisited. There's another thing that I think we've learned in this process, which is that maybe those regulations shouldn't be just about any DNA. What if the DNA is a plasmid that can replicate? Now you can slip something through that loophole and get the DNA to make more of itself once it gets in the cell. So we have the capacity today to sequence every piece of DNA that's in there.
We didn't have that back in 1984 when they were conceiving of these liability waivers. But today, the cost of sequencing has gone down a 100000 fold in the last decade. So there's no reason why we can't know precisely what type of DNA is in every single contamination event. So, a lot of things have changed since those rules were written, and they probably need some revision. So all right, let's get to the clinical implications.
What could happen to this DNA if it gets in? I'm a little bit less concerned with Moderna's only because the nature of the plasma they have contaminating has it doesn't have a few of the features that are in Pfizer. And they seem to have a better job. They're lower in DNA contamination levels than Pfizer. So if you get through the patent literature, you might understand why that Moderna actually has a patent out there that speaks to the residual DNA risks.
And they've invented technologies to get rid of it, and it looks as if those technologies work because they have less of it. But inside that patent from Moderna, it'll point out that this DNA is a risk of insertional mutagenesis, which means it can insert into your genome and cause cancer. It's a hypothetical risk. They didn't present data showing it's causing cancer in people. They just knowing molecular biology, if you put DNA into a cell and it can get to the nucleus, it can integrate into your genome through a process known as either non homologous end joining or microhomology mediated end joining.
These are processes. It sounds like anyone engaged in this type of molecular manufacturing process. It's it's a known thing that you are probably gonna get some amount of DNA contamination in here. It's known to the extent that Moderna actually invented methods to

reduce the levels of contamination from that residual DNA. So they had less of it, it appears, than the Pfizer vaccine.

But the other complicating thing here is, you know, even with other injectables that are known or could have DNA contaminants, some of those thresholds you mentioned around what we allow are based on the idea that if it's naked nucleic acid in there, it's going to degrade pretty quickly at some known rate or some rate that we can estimate. But because we're using these lipid nanoparticles to shield the nucleic acids with these new vaccines, If you have DNA contaminants in there, they themselves might be protected by lipid nanoparticles, which could hypothetically enable them to stick around long enough to do something where they would simply be degraded if they weren't shielded. Jack Mall (zero fifty seven:fifty seven): Yes. Yeah. And we've done some work to move this from hypothesis to a little bit more sound theory.

One thing you can do with these vaccines to estimate how much DNA is in the lipid nanoparticles or outside If you treat their vaccines with DNase 1, you won't see a CT shift in PCR for their vaccine DNA, which tells you that most of this DNA is actually protected from a nuclease probably inside the LPs. The other thing that we've done very recently with Uli Kammerer in Germany is she's taken these vaccines and treated ovarian cancer cell lines with them. And then, grown them in flasks and then passage them into several rounds of growth to show that the DNA persists inside these cells through several passages. So that tells you as well that the DNA is in the cells. For those not familiar with cell passaging, you treat these cells with the vaccines, a small amount of them.

A third of a dose, I think, was what she used in her case. And then you seed those cells into a dish and let them replicate a couple times to go to confluence. Then you rinse all the stuff off the cells to clean off any of the residual vaccine, take a portion of those, PCR what's in the supernatant, PCR the cells, and then put the new cells into another flask, let them grow out again, rinse them off, and then PCR the supernatant in the cells. And when we do that, we can track how much RNA is outside the cells and how much is inside the cells. And we can see this DNA inside the cells through several passages.

That tells us that the LNPs are in fact delivering this DNA into the cells. Granted, they're cancer cell lines. They're not patients because it's more complicated to do this work on patients. So so yeah. So let's let's really break this down for people.

So you've got human cancer cell lines growing in a petri dish. You put the mRNA vaccine, you dose them with mRNA vaccine, you just spray it onto the cells, you let them divide some number of times, and then you're saying you can find the residual DNA contaminant from

within the vaccine inside of these cells? Yes. And is that are they inside the cells of the supernatant and of the cells. And we could see the PCR signals in the supernatant in the cells in passage 1 and in passage 2.

And that told us that there's a good portion of this DNA that's actually in these cells. The other thing she did is she stained the cells with immunohistochemistry for Spike to see like, Okay, if it's in the cell, are they expressing? And she got the cells to be about 50% spike, IHC positive, which she's aiming for is not to have every cell transfected, but maybe half of them transfected. So those are the 2 bits of information we have. We then went and did whole genome sequencing on those cells, and that revealed some other interesting information.

The whole genome sequencing gave us about 3000x coverage over the vaccine. So in sequencing, coverage is the number of times you sequence the molecules. So at any given base in the vaccine, we had at least 3,000 reads covering the vaccine. And in the actual ovarian cancer genome, which is a much bigger genome, we only have about 30 fold redundancy in sequencing it. So there's about a 100 to 1 ratio of plasmid to the actual ovarian cancer cell line.

That's perhaps not too surprising when you think about how big the human genome is and how small this plasmid is. And when you deploy this much sequencing, you should expect to see more of the actual vaccine there than the human genome, at least from a coverage standpoint. These are only 7,000 letters long. The human genome is 3,000,000,000 bases long. But I think what was most shocking to us is that we could see that there were variants in the vaccine plasmid backbone that didn't exist in the vaccine that we sequenced that was outside of the cells.

So as a control, we sequenced the vaccine directly and then we sequence the the cells that were treated with the vaccine. And when you look at the assemblies of the vaccine in the cells versus outside of the cells, they're different. There's a fair number of variants that are only in the origins of replication in the plasmid. That tells us the cells are doing something with that DNA, perhaps replicating it and creating some I assume because the plasma DNA remnants that were in the vaccine get into these cells and persist for some number of cell cycle divisions and replications. And because this you found variants, so the the sequence inside of these cells sometimes did not match the original sequence that you found in the vaccine itself. That implies that perhaps the DNA, this contaminated DNA is being replicated in the cells. And as a natural consequence of being replicated some number of times, there's gonna be some amount of mutation. Yes. And all those mutations were concentrated in 3 regions in the

plasmid that are known as origins of replication. There's an F1 origin of replication, there's an SV40 origin of replication, and there's a bacterial origin of replication.
The only variants we saw were in those regions, which leads us to believe and if you look at the sequence depth of coverage, there's a little bit of a pull up in those regions suggesting of those things might be getting replicated by the DNA polymerases inside the cell. That's probably the most interesting evidence we have in terms of it's bioactive. Like, the only way that that DNA gets changed is if it's in contact with a cell that's changing it, Whereas the control vaccine didn't change at all. That's another hint we have that this is inside the cells because you won't get these variants if you just leave the vaccine out and sequence it alone. So there's a couple pieces of evidence pointing to the fact that this DNA is getting into cells and it's bioactive.
And now there's another biv evidence that we have here that needs further replication, because when you start talking about DNA integration, you have to be very scrupulous on ruling out all types of artifacts that can happen when you try to measure this. So we did find, 2 regions where there was spike sequence joined to human sequence, 1 on chromosome 12 and 1 on chromosome 9 in this dataset. Now the 1 on chromosome 9, 9, another researcher in Japan, Doctor Hiroshi Arakawa, has kind of sleuthed that out and thinks that might actually be an artifact of the process we have. But the one in chromosome 12, he believes is real, and has presented some additional evidence that there is evidence for microhomology mediated end joining there. There's a short set of sequence in spike that's similar to chromosome 12 that has fused with chromosome 12.
Now we have only 2 reads that are covering this out of the 30 x coverage we have. So it's it's not present everywhere, which is expected. You're not expected every cell to get, to get integrated at the same place. But we've got 2 independent molecules of Illumina sequencing that have about, you know, 60 bases in either side that prove this is in fact a fusion between chromosome 12 and spike. Now we don't know if this fusion is occurring in the nucleus or if this is some type of extra extra chromosomal DNA artifact.
And in cancer cell lines, there's this process known as chromothripsis where the since the DNA repair enzymes are somewhat dysregulated, you get shatterings of the chromosomes. And so there's a lot more extra chromosomal DNA in cancer cells. And we could have picked up an integration event to the extra chromosomal material, which isn't necessarily as persistent as something that would be an integration event in the chromosome. The reason we're presenting that caveat is that we only have one junction of the integration event covered with this evidence. Usually when you have an integration event into a genome, you should have

chromosome 12 on one side and you should have chromosome 12 on the other side and spike in the middle.
We've covered a spike human integration event on one end, and we didn't see it on we don't see the other end of it. So either we didn't have enough sequencing depth to find it or it's an extra chromosomal debris that we've picked up. But, that's another strong evidence. The DNA the only way that DNA can fuse to human DNA is if it's inside the cell. So, it's clear to us the DNA is getting transfected into cell lines, but the jury is still out on whether this integration event is something that's heritable.
It could be something that occurred in cancer cell lines only, and it's going to get thrown out in cell division. We need more depth of coverage and a lot more sequencing to confirm that it's actually a chromosomal integration event. So, you know, these these are experiments. They're cells growing in a petri dish. They're cancer cell lines.
They're not healthy human cells. We're not talking about what's happening in human beings, but of course, that's what's going to be in the back of everyone's mind is to what extent is it plausible that this type of thing can actually happen in a human being who received these vaccines. How do we begin to think about that and what does the process look like for actually determining that? Michael Osterholm (zero forty five:fifty four): I think a good This of course requires some skill sets we don't have. So we usually need to get an IRB in place and a CLIA laboratory to begin looking into that kind of work.
So we can play with cell lines. We don't have all the right regulatory structure to be working with, consented human DNA. But, there are researchers out there that are Philip Buchholz is one who's put out an offer to sequence anyone's tumor to look for this stuff. That'd be probably the place to look. I don't think you're going to find this if you just go fishing randomly through someone's genome at a random tissue per se.
I think it'd be better to focus the effort on a tumor type that's evolved post vaccination. It'd be great if anyone has had a tumor that's showed up right at the site of injection, and that's kind of an indication that maybe the vaccine is causing it, and that would be an ideal tissue to sequence. So those studies are ongoing. There are folks that are pursuing them at the moment. This cell line thing is simply meant to be a model to get us more information about what could be going on with the vaccination process as you can get in there.
It may be a proxy for what someone who's in remission may experience post vaccination. You kind of have to remember that we are always cancering. It's when our immune system falls down that the cancers emerge and to be problematic. Normally, our immune system is clearing ourselves of cancer cells daily. And you need you need sort of a a multiple hit

hypothesis here to weaken your immune system and increase the mutagenesis rate in order for these cancers to outpace the immune system.
So you be able to view the ovarian cancer cell line as a proxy for what might happen to an ovarian cancer patient who's in remission post vaccination. Can any of their cancer cells reemerge post vaccination? But it's not a person, and it's not meant to We're not here to say that this is the perfect model for estimating. It's the right thing is to eventually do this in humans. But before you go and do this in humans, you need to work out some of the methods first on cell lines before you just start doing fishing random fishing expeditions inside patients' tissues.
It's just a this is that step of the process of sort out the techniques and the methods we need to do this so that, when we do approach patient tissues, we can do it very efficiently, and we're not trying to learn on the fly with patients' tissues. So it sounds like tell tell me if this is accurate. It sounds like you would say it's been demonstrated convincingly and replicated that for these mRNA vaccines, there are remnant pieces of DNA from those plasmids that they use in the production process that make it all the way through and into the final product? Yes. Yeah.
That's been replicated by many labs. The work that I just spoke to you about regarding treating cell lines with has only been done with ourselves and Uli. I've heard of one other lab that's seen it as well. Didier Ryot called us saying he's done similar work, and he's seeing the DNA get into the cells, but neither of us have published anything more than a substat. Jason Brett (3six forty six): So that work, tell me if this is a fair summary of that work.
That work shows that in principle, it's possible for some of this remnant DNA to persist inside of cells and be replicated, a number of times to potentially mutate and potentially even integrate into the genome of the host cell. And that means it's at least theoretically possible that could be happening with the mRNA vaccines in people, but it does not prove that. It does not prove it. And I also caution that so the PCR work has been replicated by many others independently. The cell line work that we just spoke about has not yet been replicated by other people.
So it's much more nascent. There is some, you know, word-of-mouth that we've had, you know, sharing with other colleagues on this that they've seen it as well, but all of this needs to get kind of summed up into a paper that we'll that we're going to shortly submit. So, there's more replication that's needed on the cell. Treating the cells with a vaccine and what happens is is really fresh fresh territory. What does all of this start to say about you know, this is new technology.

These are mRNA vaccines different from vaccines that have been historically used to new type of technology, due to the COVID pandemic. And we all probably remember the early days how much uncertainty and how much panic there was. You know, we wanted to rapidly deploy technology like this as quickly as possible. How does this start to get you thinking about, like, weighing the pros and cons of, okay, we want to we want to scale up and rapidly deploy this type of new technology, which looks promising in many ways against the fact that it's new technology, and we don't know all of the little quirks and things that are yet to be discovered. Yes.

So, maybe COVID isn't the best analogy for this just because the IFR, and it is so low in kids that I don't think they just necessarily warrant this type of risk. But there's terminal diseases out there that perhaps I mean, I know Alnylam's out there with they have an RNA that they use. It's not it does not come from a plasmid. It's a very short interfering RNA, which is like maybe a 20 to 30 base pair piece of RNA that's chemically pure. They put into LNPs and they infuse into people over an 8 hour drip under immunosuppressants.

So that's a very different approach of using RNA. And I can't speak to how well it works. I've just not heard of these really strange adverse event profiles that we're seeing on the COVID vaccines. And that's also being utilized for a terminal disease, right? So they've kind of found a disease where the risk benefit ratio is obviously very different than this respiratory virus. Their administration is being done much more carefully through infusion with immunosuppressants over long periods of time. When you turn to the COVID vaccines, we have a a large class of people that don't need them, a large class of people that already have natural immunity, and we're taking very similar gambles without having the same precautions in place. We're not infusing these over periods of time with immunosuppressants like they're using with these other drugs. So they're very different. I know people want to like cast stones at all RNA and kill it forever.

I'm not of that mindset in that there are applications that don't have these manufacturing defects that seem to be in the marketplace, that don't have a VAERS explosion going on that I'm not willing to, like, condemn because there might be a use for this in some discrete circumstances in healthcare. So, But in terms of COVID, I'm just perplexed at the biology here. I don't understand why we're trying to build immunity through injection. Most of these viruses enter through your mucosa, and it's very hard to get mucosal immunity through injection. You build antibodies in the wrong compartment of the body.

So so maybe they protect you if the if you had bueremia and the virus went everywhere and into your bloodstream. They then they would turn on, but by then, it's kind of too late. You

really want respiratory immunity in your mucosa, not not, not in injection. So I I think it's the wrong platform fit, if you will, for, for respiratory viruses. Set aside all the purity issues.
If they perfectly cleaned everything up, you have to ask yourself, Is this the right way to stop a respiratory virus? And it has to be really, you know, I think a very virulent virus for you to consider it given, the unknowns that are involved. So, you know, I'm not here to condemn the whole platform. If they can clean up the manufacturing and find some type of use case where the risk benefit equation is very different than what we've been presented with, then they may make sense. But I'm having a hard time finding that in anything related to a respiratory virus.
What, are the vaccine manufacturers, like, doing this type of work to clean up that process? Has the work that you've described that you've done prompted them or regulators to tell them that they need to do some more stringent testing and just streamlining of this process? I don't know that it has. I mean, there is, the Prep Act gave them additional liability protection. My understanding of the Prep Act is it extended the liability protection to beyond the pharmaceutical manufacturers and even to the administrators of the products.
So the nurses and doctors who are administrating these things have some shield over them as well. So in absence of liability, I suspect they were not financially motivated to do anything. And the signaling we're hearing from the FDA is that there's nothing to see here. They the manufacturer claimed they tested this. We trust that they did, so we're going to take their word for it.
I mean, what I'd love to see are the regulatory agencies actually perform some PCR because I think when they do that, they'll recognize that the numbers are higher than what the manufacturer promised them. I mean, the highest lot we've seen yet came from Germany. It was at a CT of 13. So for those familiar with PCR, you may have been called positive, at a CT of 33, for the virus outside of your nose. That's a 20 CT gap by a million fold gap.
So there's a million fold more of this contaminating DNA being injected, then you might get called positive for remnants of virus that are outside of your nasal mucosa, not inside per se. So that that's a that's a large difference, to be concerned about. So they're probably thinking the CTs on this are out in the 23 range, which where they should be if they're below the guidelines, 20 to 23 range. So I think they're putting a little bit too much faith in the manufacturer's measurement of this. There should be independent measurement of this, and the techniques that are being used should be published.
This is one thing that has concerned us is the techniques we see looking through the EMA documentation shows that they're allowed to measure the DNA with qPCR, which is very

stringent and under measures it even according to Moderna's own patents. But then when they go measure the RNA, they can use fluorometry, which will over count the RNA. We've put some of this in our preprints, but the fact that they're measuring these things with 2 different yardsticks shows you there's a game going on because anyone who's done PCR or done a COVID test for that matter knows that you can measure DNA or RNA with PCR. So if you're asked to be measuring both, you should be using the same yardstick for both, not playing this game of I'm going to inflate the RNA with one technique and then use this PCR method to deflate it in another technique so I can slip through the regulations. So there is there is a bit of a of a game going on as to how they're being held to these regulations, so I'm not surprised the regulators are confused by this.

They may not be, you know, alerted to the fact that the techniques that are in play here give you vastly different, quantities of RNA and DNA if you if you give them the freedom to cherry pick the tools. Let's talk about spike protein itself a little bit. So the intended way these vaccines are supposed to work is the mRNA encoding the spike protein gets into our cells. Our cells use that to make the spike protein itself from that mRNA. And then they are presenting that spike protein to the immune system and that triggers an immune response.

And that's how we generate immunity from these mRNA vaccines. Last time we spoke, we were talking about how, when you look at some of the data that's out there in this documentation, the Western blot data, it sort of looked like when you look at it closely and you know how to look at the stuff, there wasn't just native spike protein being produced, but these these bands were smeared, implying that there were variant variants of the spike protein being made of some frequency. And we were speculating that might be due to this frame shifting thing that can happen with this modified uracil, a nuclear base that's used that we talked about earlier. And since then, and at the end of 2023, there was a paper showing, in fact, that you do get this ribosomal frame shifting. Can you summarize that for people? What exactly did they find there? Jack Wolfson (3five zero six): Yeah. So these bases, when the ribosomes are going across reading these bases, they read them in sets of 3. And, you can imagine if you change one of those bases and and put a methyl group on it, that it may confuse the ribosome's capacity to translate that codon into the right amino acid. And that's, in fact, what that Mulrony paper is showing is that, in particular, when you have 3 pseudo urines in a row, the ribosome gets confused and slips on it and goes off by a frame.

And then it starts reading the RNA transcripts one base out of frame, which is entirely different language, and that and makes spurious peptides after that point. So that is what the Mulrony paper demonstrated. I think that's half of what's going on. The other concern that

can happen in making these RNA templates is these same bases that are difficult for ribosomes to read are sometimes difficult for the polymerases to read. And, there's something known as template switching that has gone on, and that's at least in in Moderna's own documentation.

So they're they're very scrupulous at looking at how much double stranded RNA is in these things, and they even published a paper showing that they mutated T7 polymerase to make it to stop doing this, this template switching. So what is template switching? Everyone confuses that with frame shifting. But template switching is when the RNA is getting made, and the enzyme falls off, and it's not completely made yet. And then the strand hybridizes to another strand and it starts copying in the wrong place.

All right? So there's 2 forms of template switching. There's trans template switching where the template lands on a different part of the DNA molecule and primes off of that, and it's therefore making a chimeric template. And then there's cis template switching where the RNA loops back on itself and goes the other direction. That cis template switching is called, like, loopback extension or loopback extension or loopback, polymerization.

So Moderna has this interesting paper showing that when they mutate T7 polymerase, they can eliminate a lot of this template switching. The problem is that paper came out in 2023, and it's not clear to us that they had that operational when they were making the vaccines, neither Pfizer or Moderna for that matter. So what that can mean is that you can have some RNA templates that are not correct. Part of it's correct, and then hopped onto another piece of DNA and hybridized there and started making more RNA that's not correct, a chimeric RNA, if you will. Or it looped back on itself and started making more RNA.

Both of those mechanisms can create RNA that's longer than the expected length, And that is something that we see in some of their plots when they look at RNA integrity scores. They run these things on electrophoresis system. You should see a really discreet peak at 4284, which is the length of our mRNA. But in fact, what they see is a smear before and after that RNA, which is a sign that there's longer transcripts in there and shorter transcripts in there. And those could lead to other types of proteins, if you will.

So I think Mulroney did a great job showing the frame shifting is in fact happening with the ribosome and translation, but there's another layer deeper than this that I think has been harder for people to pin down, which is what's the degree of template switching and double stranded RNA that's being made in this process? Now in the case of the Pfizer vaccine, I'd be more worried about it because the Pfizer vaccine, the opposite strand on the Pfizer vaccine doesn't have any stop codons. So if you do get any loopback RNA extension on Pfizer, it's going

to make a chimeric peptide for a very long time because that piece of DNA has no stop codons in it. So if you get a loop back mRNA and you feed it through the ribosomes, it's going to keep making amino acids until the end because it has no stops built into it. So there's a particular, I think, unique issue for Pfizer with template switching because their codon optimization eliminated all the stop codons in the reverse strand, And and that means that there's an open reading frame that's undisclosed in their vector.

That is not presented to the regulators. The regulators demand that you have every open reading frame disclosed, in in a product like this, and that reverse or has been omitted from any disclosure to the EMA or the FDA. So so the Mulrony paper that looked at ribosomal frame shifting, that was in vitro. Right? It wasn't in it was showing that this happens in cells.

I thought they actually had patients in there. It was a small number, and I thought they were pulling that out of maybe maybe there were patient cell lines that they derived to to say that. It's a good question. I gotta go back and read that and Oh, no. Hold on.

Yeah. I have the paper right here. Ribosomal frame shifting in vitro and that cellular immunity in mice and humans to plus one frame shifted products from the Pfizer vaccine mRNA translation occurs after vaccination. Yes. There you go.

Okay. So so you had 8% or something of the proteins that they were mentioning in there were were frame shifted. So so okay. So what what what's the punch line here that when these mRNA vaccines are used, you're producing native Spike protein, but you're also producing a small, maybe 8 ish percentage of other variants on that spike protein that are not exactly the same. Yes.

And and we don't And is that a question? Yeah. Okay. So it's a question mark if they're harmful or benign or whatever. Yeah.

This is true man. Hey, maybe that's, you know, maybe those aren't harmful. But but I think to your initial point was how is this different from manufacturing a peptide and injecting a traditional vaccine? You're not exposed to this risk because they presumably purify the wrong proteins out of any such thing that would occur. There's no pseudo uridine involved in those things.

So the frame shifting should be a much more frequency. So a traditional antigen that you inject wouldn't have this. You're not playing games by trying to camouflage the RNA, get your cells to make it, trick them into making it, and and then exposing the cells to, you know, mistranslating these things. Yeah. So I guess just an an inherent part of using mRNA to make your antigen, to make the protein that your immune system is going to recognize, is that an inherent part of this process in our cells is is the there's just noise.

Like, there's gonna be some noise. There's gonna be some, infidelity that the system has, and how much it happens is is gonna depend, and whether or not it actually matters in the end is still a question mark, but it opens up this possibility that things other than what you intended are now gonna be inside of your cells. Yes. Yeah. I think that's something I've learned in building DNA sequencers is that when you modify a base, all bets are off.

I mean, it changes the way the sequencers read them. It changes the way the ligases handle them. It changes the way the polymerases hand them. Like a simple people will be like, Oh, it's just a methyl group. No way.

Changes all the dynamics in terms of the melting temperature of those bases. It's a complicated system, And nature has evolved these little signatures on bases for a reason because they hitch onto them to perform certain biochemical events. So decorating an mRNA with a 100% of these different bases is not something our cells have seen before. So, we're in an unknown territory as to how the cell's gonna, respond to those things. You know, when you think about all of this molecular biology and all all of the details and complexity here, how does that influence how you think about the landscape of, you know, potential vaccine induced, side effects and things like that?

Like, the people argue about this stuff because it's a highly politicized area. You know, you're seeing different things at different rates and different populations. You know, obviously, people have talked about the the myocarditis issue. How do you think about all that stuff in the context of what you're learning as a molecular biologist? I'm a little bit biased here because I spent 5 years doing mitochondrial sequencing.

And so what really unnerved me about the Pfizer vaccine is that after the stop codons in the mRNA, they have a 5 prime UTR, an open reading frame, and then a 3 prime UTR. UTRs are these untranslated regions. So they don't they shouldn't get turned into amino acids, but they are in the transcript of the RNA. So the the ribosome is supposed to start the start codon, an AUG, like a methionine, and then keep translating until it hits a stop codon. Now in the case of Pfizer, they put in multiple stop codons because they know when pseudouridine is in a stop codon, it sometimes doesn't behave as a stop codon, and it can frame shift.

Fernandez has work showing Fernandez et al is a paper that we referenced in our preprint with Peter McCullough showing that when ribosomes read sudugiurdines, they sometimes frame shift across stop codons. Now what's So what's after the stop codon in Pfizer is a mitochondrial sequence, a human mitochondrial sequence. So if we're making any and if we're slipping through that that stop codon and making any human mitochondrial peptides, I don't know what that's gonna do. I don't know if that's gonna make the spike localized to

mitochondria. I don't know if it's gonna gonna trigger some kind of immune response against a mitochondria, which I don't know how that would happen.
They're intracellular organelles. But to me, that's a red flag only because when I read about a lot of the people who claim to have vaccine injury, I see things I saw in the mitochondrial space. I see POTS. I see dysautonomia. I see brain fatigue, or brain fog, and chronic fatigue syndrome, and all of these things that were showing up in the mitochondrial disease space. So, I'm really curious why they have that. They claim they put that mitochondrial sequence in there to make it express better. Once again, they're trying to step on the gas to make it last as long and and and as powerfully as possible, but that's not always the right approach in pharmacology to have, you know, everything on at full blast. So I would be worried that there could be chimeric spike human peptides in there, which could create autoimmune issues. So, I don't I don't have any proof of this.
I just see the spectrum profile of patients out there that have that are vax injured, and it seems to be very mito based. And then I see that there's mitochondrial sequence inside of the Pfizer vaccine and wonder what happens when you frame shift shift into that stuff, and does it does it create these, these chimeric peptides? Can you talk a little bit more about the persistence of the spike protein? What does the literature say out there in terms of how long spike protein produced from the vaccines is actually lasting in the body? And is that Yes.
Is it what we think is it what we thought it was originally? Yeah. It's not what we thought it was originally, but there's several there's 2 different avenues here. There's the nucleic acid longevity, which right now the data we have shows it's shorter than the actual protein. And some of that could be measurement technique and the number of studies that have looked for both.
So on the vaccine front, we've seen Cryosin et al. Demonstrate it in the heart for 30 days. We've seen Castruta show it in plasma for 28 days. Now we've seen Hannah show it in 2 different studies from Hannah et al. In breast milk, 5 days or more.
There's a lot of critiques that these numbers are such small numbers that they don't matter, but I have some sub sects that go through those numbers. Some of the issues with the first hand of paper was the PCR test they had only had a LOD at 440,000 molecules. So it really couldn't see it out to 30 days because the PCR test was several orders of magnitude insensitive for the job. There's another paper recently, I'm going to murder in the last name, but it was in placenta. And they demonstrated 1 patient that was vaccinated 2 days before birth and one vaccinated 10 days before birth and they picked up, RNA in the placenta.

Now most of the papers looking for RNA right now aren't really splitting hairs between RNA and DNA. So we don't really know if this is an RNA or DNA issue because when you do RT PCR, it amplifies both. And most people, I think, were assuming these vaccines had no DNA in them. But you have to go back and revisit those studies with some nucleases present to sort out whether it's RNA or DNA. So that gives you a window of about 30 days, okay?
Somewhere between those studies went from 2 days to 30 days, on the nucleic acids. When you start getting involved with the spike protein itself, we have the Bansal paper that showed that it was in exosomes. I think that was 4 months out and, forgetting the author name, but a 187 days was the longest one I think I've seen, on showing spike protein in, what's the name of that? They might come to me later, but there's a, there's a I think I have a link to it on my stack for, for the spike protein. So we're seeing the protein sit around longer.
Now a lot of people will point to, well, shouldn't the immune system be clearing out the spike? Why is it sitting around for so long? And there there are a class of cells in the body that are immunoprivileged cells, like stem cells, that your immune system won't clear out. So if the LNPs get to the stem cells, that could be one source for why there is spike that is evading, clearance. And so yeah.
So so if the LNPs get to those cells, how might that happen? So, you know, I've heard people talk about, well, we're getting these injections into our arm. They're meant to be like intramuscular. But, you know, some percentage of the time, right, it's going to hit an artery or a vein and get into circulation. And I guess the concern would be if and when that happens, and it's inevitably going to happen some fraction of the time, that in those people in which it happens, those LPs are flowing all over your body and maybe getting to other places.
Yeah. That that's that's the best theory I've heard is that, yeah, accidental because and this would probably explain why, like, not everyone's getting sick with these vaccines. There seems to be a sort of stochastic nature to it where a lot of people have taken these and they're fine, they've experienced no issues. It's just a subclass of people. And maybe there's another confounder here, which is certain lots from our study of the lots, they vary.
We've seen stuff that's varied like 10 CTs. There's a thousand fold variance in, in the amount of DNA contamination that we've seen just surveying maybe 30 or 40 lots. So you could have people who have bet lots that are getting a lot more of that contaminant. You could have people on top of that that, it's, you know, it's staying in the arm, it's contaminated, no big deal, gets into the circulation, goes to all the cells, and now you have a transfection experiment with contaminating DNA across all the cell lines. So there can be a combination of these things.

It gets very complicated, but I do believe that there is some sarcastic nature of the injury, Right? Not everyone is hurt by these things. So we have to keep in mind that maybe it's only the people that get hit in an artery where this thing can cause us problems. And some people may get hit in an artery with a fairly clean lot, and it's not an issue either. So there's You can't use the fact that there are billions of people injected to say that there aren't people getting hurt by these.

I get that all the time online that these are safe because billions of people smoke cigarettes. You know? And it's like, no. If you're not counting the injuries correctly, that's not a fair way to go about assessing the the risk here. What do you so the myocarditis, pericarditis issue, my read of that literature is that, it depends on demographic, right?

There's an age and sex dependence here. And basically younger males are at higher risk. The absolute risk is relatively low, right? It's like 1 in several thousand males under the age of 20 something will get myocarditis, but it's much higher than chance would predict. What do you make of the age and sex dependence there in the first place?

Why would something like this depend on the demographic you belong to? Jeremy Grantham (zero zero six:fifty six): Yeah. It's a good question. I just heard a podcast from, his name is and any Coker on on Jay Bhattacharya's podcast, I think the illusion of consensus, where he was going through some of the studies that were looking at myocarditis caused by COVID, which he wasn't very convinced of. The studies had a a lot of flaws in them and he walked through some of those flaws, but the ones from the actual vaccine have some real hard clinical evidence through autopsies and through looking at with MRIs and looking at troponin levels. I don't know why it's hitting males more than females at that young age demographic. He was bringing up androgen and other types of, you know, hormonal issues that might be at play. So that those are that's not my field of expertise, so I can't comment very, very effectively on that, but I I I do that was a good podcast that walked through that. That's worth anyone who wants to dig deeper on it. When you're doing, like, the research that you described, in this area, you know, given how much controversy there is when it comes to some of this stuff, how intertwined this whole area is with politics and stuff.

How has it been trying to publish work? And I don't know. Is there anything interesting going on there? Is it is it more difficult to publish work? I mean, it definitely seems like right there's there's lots of motivating motivated reasoning going on.

There's a lot of people who want to prove this, who want to prove the opposite. What's what's it been like doing the research and trying to get it published? So we haven't been very successful in getting it published. So that that that all the, The first one we put out with Peter

McCullough that described this frame shifting had 2 reviewers approve it, and then the editor swept in and killed it in some very curious way. But lo and behold, the frame shifting turned out to be pretty well forecasted.
Okay. So hold hold on. You submitted a paper. 2 reviewers looked at it, the normal peer review process. A couple other scientists anonymously review a paper.
They said, go ahead and publish it. And then after that, the editor, the person above them came in and said, no. We're not gonna publish it. We're not doing it. Yes.
Now What was the reason? It was at Hindawi, and I they didn't give us a good reason. That's what was really frustrating. And I did notice after, we received that, there was a notice that Hindawi was like retracting like 500 or 1000 papers that they had gone through some investigation. So I think they were under probably extreme scrutiny at that time, going through this process of of, you know, retracting 500 papers.
So we may have hit them at a bad time, where they're like, alright. We're just not taking any risk right now because we're in the process of doing all this retraction, but they didn't give us a reason for it. They just they just said, no. We're not we're not moving forward with your with your publication. So we ended up saying, alright.
It wasn't as clear as that, to be honest. They they were they were like they just kept dragging it out saying, we're not we're not we need an extra review on this. We've we've graduated your paper to go to another tier of review because, you know, whatever for whatever reason, and we just got tired of them and said, listen, this is taking too long. We're going to go somewhere else with it. And that's kind of how things were left with NDAU.
So that was that case of and that wasn't even a paper with a whole lot of experimentation. We just did some bioinformatics analysis on the number of like quadruplex Gs that showed up in this and a couple other RNA fold issues that were somewhat interesting. I think the interesting one interesting detail in that paper was that I don't know if you were familiar with this Andy Fire paper that came out recently looking at obelisks, which is So it's really fascinating work that they found. They went combing through the SRA looking for double stranded circular RNAs. And I work in that field with viroids or double stranded circular RNAs, but they don't code for anything.
And he found a bunch that were about a 1000 bases long that actually have an open reading frame, and they're circular, and they're hairpins. So they're a new like chemical entity, if you will, known as an obelisk that they coined. But that paper led me to one that shows that the mRNA folks are designing their codon optimizations to be very rod like because those are harder RNAs to destroy, and they last longer. And I hadn't known I didn't know that when I

wrote the paper with McCullough because I noted I did see that they made all the RNA codon optimization made them hairpin a lot more. And I was just shocked at the secondary structure change.

When you put the codon optimized RNAs through RNA fold, you see all of these hairpins that aren't in the actual virus. And then that Andy Fire paper came out linking to a group that said, No, when we code and optimize for mRNA vaccination, we try to turn them into rods because these hairpins protect them for degradation. So but it was mostly an insuloc analysis with Peter McCullough and a bunch of literature searches around what could go wrong. So there wasn't a whole lot of stuff to critique other than, you know, we don't like the collection of citations you put forward here. So most of the reviewers were like, yeah, this looks good that you've all these are real citations and, you know, the paper sound.

And then I think, Kendawy got stuck in this, okay, we're in a retraction mode now of, you know, retracting a bunch of papers. We're going to put all papers that are controversial through a secondary screen again. So I think that's where that one got caught up. But since then, this work, having had that experience, we just went with the approach of put this stuff public as quickly as possible and do the extra work in the lab to make it easy for someone else to reproduce it. Like it's one thing to make a paper knowing that someone is going to peer review it without a pipette.

It's an entirely different matter to say, No, we need to actually build additional assays so someone can pick up a pipette and reproduce this. That's a whole another month of work, which we did on this. When we put out the work with sequencing the vaccines, we didn't stop at just putting the sequence public. We went and designed quantitative PCR assays that targeted 3 parts of the plasmid, fine tuned all that stuff, put the primers public, and that enabled, you know, many other labs to just pick up the primer sequences and check their vials for, like, $100 instead of having to sequence the whole thing and and and sort it out. So if you're going to go this route of, I think, being very transparent, you have to put more work into not necessarily thinking someone's going to just read the paper and double check it, but anticipate someone to have to replicate it at the bench and do the extra work to make assays that afford that, and make it easy for replication.

Because you're at that stage not relying on reviewers blessing this reading it. You're relying on the field replicating it at the bench. And I think that's a faster way to go just because I don't really care about peer review, I care about replication. And the moment people start pipetting, you get better feedback. Like when Philip did this work, he pointed to a few things that we missed.

And when Xin Li did some of the work, he taught us things. And David Speaker taught us a few things, right? So the pipetting is the real review and that's where we actually sharpen the pencil quite a bit. So it is politicized. It is difficult, but I'm losing faith that the system we have isn't like captured in consensus building and that it's hard to publish something that bends the narrative.

The system is very much narrative reinforcing, the current peer review system. And if you're outside of that, you're probably not going to have a good time at peer review. It's probably going to take a year, and you might want to consider going with a preprint with that has a hell of a lot more effort focused on how does someone get the lab reproduction done as cheaply as possible. In your mind, like, what what like when you're reading papers, if you're looking at a preprint versus something that has become, you know, an official, quote unquote, paper that's gone through the peer review process and gets into a major journal. How much does that how much does that matter to you?

Do you do you think the the pre preprints are are less worthy than the ones that make it through peer review? Do you think that doesn't matter so much at this point? I think what happens is is for me, I'm if it's in my field, I go right to the methods section and look at the conflicts. And if I can understand the methods and the results and the conflicts look clean, that's all I care about. I usually ignore the abstract and conclusions because those are usually marketing things that you put forward to the journal to make them interested in publishing it. Like, this is a story that has great significance for x, y, and z. And they're oftentimes not necessarily evident in in the results. Right? People can look at results and interpret different things out of them. So if you're a set of authors who is in the world of like, these vaccines are safe and effective, you will see a set of data and be like, this shows that they're safe and effective.

Another person could see the same data and be like, well, I'm not so certain. I I view it differently. Right? So conclusions are always there's always a subjective nature of conclusions. The author's interpretation of the data is embedded in the conclusions, whereas the results of the methods themselves really tell you everything you need.

So in my own field, I don't discern between whether something's been published, whether it's a poster, or whether it's on a preprint because I can decipher it. When I get outside of my lane, I then have to rely more on other experts. And I'm easier fools, and I tend to read into the abstracts and the conclusions a little bit more than I probably should. So those are I think the most important thing if you're outside of your wheelhouse is that you scrutinize the

conflicts first. And if there's a lot of conflicts there such as this is funded by Pfizer, and I'm like, Okay.

I don't have time in my day to read a Pfizer paper. I'm going to move on to one that doesn't have conflicts. So I tend to put a lot of weight on the conflict of interest section only because I've seen so much of that in COVID where the results have sponsorship like that, and they're just polar opposite of what I'm seeing from like physicians on the ground and what they're telling me, that I can't take a whole lot of faith in the peer review system, giving faithful information in that direction. And I think it's more of a concern for me when the sponsors are very large financial institutions than small ones, because they have the purse and the capital to actually advertise in that journal and to influence, perhaps, the way the editors are behaving. Many people may not know this, but the way the financing of peer review behaves is the people who want to publish give the journal about $3,000 hand over their copyright of their material to the journal.

The journal then makes money not only by receiving your $3,000 but by also doing advertisement for folks like Pfizer inside the journal, and then reselling access to the copyright of those things to Harvard and Stanford and what have you through If they put these things behind a paywall, there's like a $35 to $50 fee for someone to download the paper. So there's a risk there that the intermediary, the oracle, if you will, running the journal can be influenced by their advertisers. And I certainly think that's what's happening in the mainstream media. It's probably less obvious in the actual journals themselves. But in mainstream media, there's like every single basketball game I watch now has Moderna on the floor and brought to you by Pfizer, and they never say a negative thing about the vaccines.

And in the journals, I think to some degree this may be going on as well that they are very pro vaccine. They've always been pro natural origin of this as well. So there's been a lot of protectionism of a particular narrative through the journals that I don't think is really welcoming of people who have counternarratives. So we're trying to break that. There's a couple of people I know that are trying to build a peer to peer review system where it doesn't have a journal involved.

It's just a capacity to publish, and everything gets etched onto blockchains. And, you recruit reviewers without a journal. We'll have to see where that goes. But I think that's a better system just because I do think the review system since the review the people who are adding the most value to a review aren't getting any of the rewards. So there's no reason for me to do a peer review.

I don't get paid for it. It just takes away from my day. And so the only time that I find people jump into peer review is when it's in a field and they're worried about competing work obliterating or scooping theirs. And not that many people are going to be not many experts can you get a lot of time for free, and that's what the journals rely on to getting experts to come and review this stuff for free, Then I think the incentives are all broken. I think if you put a put a bounty out there for reviewers and it was very transparent, you'll get people to instead of driving Ubers on on on weekends, PhD students can be reviewing papers and getting paid better than they might be gaining an Uber salary, if you will.

So I think we can change peer review if we put in market incentives in place, and turn it into a more of an Austrian economy, if you will, where there is pricing signals in peer review, and you can get platinum review, gold review, bronze review. You can get fast review, slow review, you know, 5 reviewers, 2 reviewers. Right now, it's a very one size fits all review system, and there's no urgency involved. I have a substack from just this weekend actually about a peer review in the philosophy space that I went through that took 3 years. And it's just a total train wreck if you read through it all.

There's no motivation for anyone to actually review things quickly. So I encourage people to read that if they want to get an understanding of how that sausage is made, but, I think it's time to disrupt it. We saw too much of this in COVID where SERGIO SPHERE came out, Proxima Origins came out, all of these papers that massively disrupted, the patient access to hydroxychloroquine came through, and they were later shown to be frauds. So, the system doesn't have a very good reproduction rate, and reproduction is where we should focus. Did you see, what happened recently with the FDA and some of the ivermectin content they produced?

What happened there exactly, and what's your take on it? Doctor. Whitfield (zero forty five:fifty six): well, they did step out of their lane. They started mocking people using ivermectin, and it's not FDA's charter to prescribe medicine. Right?

Even with physicians, they get nervous prescribing medicine to patients they've not met or looked at. It's frowned upon. Right? You should at least have a telecall, a telemedicine, and poke and prod the patient a little bit. But, the FDA started saying Ivermectin is We're not horses.

Stop it, y'all, on Ivermectin. And so you look at that behavior and like they are becoming a marketing engine for the vaccines. Why would they pick a fight on which product that they want to support or not support? You know, Ivermectin is a Nobel Prize winning drug that's already been through the FDA, but it's no longer really protected by any IP. So it doesn't have

it's unlikely anyone was going to put ivermectin back through the FDA process and pay them again.

I mean, the FDA operates off of the PDUFA Act where about over half their budget comes from pharmaceutical companies paying them to defray the costs of regulation. So unless you're in their pay stream, they're probably not protecting your drug. And Ivermectin is something that fell off patent, and no longer really has a protector like that or sponsor, if you will. So they seem to be shooting down generics to part the Red Sea for the people who are paying them for the most latest and greatest drugs, which are, of course, the most dangerous drugs because we know the least about them. So it kind of works against generics in many ways.

And I think that was a perfect example. It's good to see Mary succeeded in getting them to reverse it. I'm only worried that they'll do it again, right? They got a slap on the wrist. They have to take their posts down and a little bit of a humility session there.

But they're not necessarily if it takes 3 years for you to reverse something like this, they're gonna do it again, and then we'll have 3 years of the courts fighting them to stop doing the behavior in the future. So I'm not convinced what, although it's a victory, I I'm I I don't think what came out of that is gonna prevent the behavior from reoccurring. You know, given how much controversy a lot of the stuff is embroiled in, you know, how politicized everything to do with COVID and vaccines and ivermectin and other drugs. How it's what a mess it's all been.

On the Ivermectin issue, when you step back and you look at all the evidences out there, there's obviously right?

There's a lot of good and bad studies published. Some of them wanting to prove one side of the equation, some of them wanting to prove the other. What's your general read on on the truth regarding ivermectin? I'm probably biased because I follow Pierre Kory and read his stack quite a bit, And it looks to me that it works. Now I'm also coming from a medical cannabis world, right?

Where I'm like, it's none of anyone's business what drugs people take. If they think it's going to help them, let them take that. And so this is one of those scenarios where they got in the way of a drug that won the Nobel Prize in medicine, and it clearly has low toxicity. Otherwise, they wouldn't have proved it for all the other things they use it for. And they were able to railroad it in favor of some other therapeutics that had had better royalty streams going into the government.

Right? There's a $400,000,000 royalty stream, going into the NIH for Moderna. I don't know how much is coming in from Pfizer, but I'm assuming Pfizer's probably going to be paying them

eventually as well. So there's, you know, the government institutions are all cheerleading for the thing that probably fills their coffers the most and trying to shut down anything that can reverse that or challenge that. So I I don't understand this.

It was never a crime to to put drugs off to use drugs off label until COVID. Right? When you say you think that Ivermectin works, what what exactly does that mean? Like what dose used for what purpose? In what way does it work?

So yeah. So the most people who are using Ivermectin aren't using it alone, and a lot of the studies that attacked it used it at doses that were inappropriate. So they're usually using it with like a Z Pak, maybe zinc, and ivermectin, like at least those three things. And they're showing I think the most recent one I saw was somewhere like in the 70 or 80% reduction in mortality with Ivermectin. And now it has to be given early.

Some of the studies would test this in hospital, at least with hydroxychloroquine. They pulled that move in hydroxychloroquine where they tested it after only they were blue in the face and practically dead, and they gave them a bunch of hydroxychloroquine. And it's not going to work at that stage. So there were a lot of trials that looked like they were set up to tarnish this with doing all types of games with the placebos and the controls and the dosages. I think the last thing I read about Ivermectin, it was somewhere around 150, what is it, micrograms per kilogram or something.

So I'm not the best person to quote on like the optimal dose here. I'd point people to Pierre's work. He's got a whole book on this. But, the few papers I've read that he's put out and his substacks have all pointed to the defects in the trials that we're trying to take this down, the connections to pharmaceutical sponsors, and then the less conflicted trials and the results that they saw, and it seems to lean in the direction of this working. And the fact that we have to do all of this at all seems ridiculous because they it was legal to to prescribe off label drugs for for this before the pandemic, and then we had to do all these damn trials just to fight over whether they could have their rights back.

So I get a little bit frustrated because I think you have some probably some similar background in the medical cannabis field as well where these products are going to be hard to get clean trials on. We have entire government agencies trying to take them down like NIDA, that likes to do studies that demonstrate abuse of drugs. And so it's hard for them to ever admit there might be a benefit in such a study. But we've got lots of patients that are demanding access. They believe it works, and that's that's where the I think the debate should end.

But it seems like it has to get politicized, and there has to be a fight over who gets to collect the revenue stream of various medications. I wanna switch gears for just a little bit while I

have you. You know, thinking about cannabis, you mentioned very briefly at the beginning, something important that's happening in that world, which is this infection, which is decimating cannabis crops. What is this thing, and what do we know about it? Yeah.
So this is Hoplatan viroid. It's a 256 letter genome, which is remarkable. It doesn't code for any proteins. It's a circular RNA that when it gets into a cell, since it's circular, it can replicate with rolling circle, amplification, but it needs the host polymerase to do that replication. And then once it makes a long concatemer genome, the folded RNA oftentimes acts as a ribosome and chops that RNA up.
I don't think that's actually happening with Hop Latent Rhino. I think that's a different mechanism. Something else in the cell is probably chopping the genomes back up and folding them back into circles. But anyway, it tends to a lot of plants it tends to sit in the roots. And then when the plants go into flowering, it travels all over the plant and reduces the yield about 40%.
There are some plants that seem more resistant to this than others, or tolerant, I should say, than to others. So we still don't fully understand the biology. We think what's happening is the RNA has certain sequence homology to about 20 different genes in the cannabis plant. And through a process of RNA interference, it hybridizes that RNA and that RNA gets clipped or diced and cut into pieces. And so it's down regulating the expression of certain genes that also enable its capacity to replicate.
So that's kind of the mechanism we think is at play at the moment, and there's no cure for it that we know of yet. People are just if they detect it in a plant, they kill the plant. So there is a lot of PCR going on in the field where It's actually a very interesting economic use case because a grow can put in a PCR system and start running PCR and see the economic gains of cleaning their grow and getting more yield in like less than a year, which is quite different than the kind of sentiment that's been left in the field with PCR for COVID where we were probably quarantining far too many people and everyone has a bad taste in their mouth over it. But it is a prime example of being able to demonstrate that, yes, PCR can contract down pathogens in in such a way that the cost of the PCR is more than made up for, the reduction in the pathogenesis of the disease is trying to is trying to sleuth out. How do growers detect this and quarantine and and get rid of it?
Yes. Most aren't quarantining. They're just if they if it's hot, they get rid of it. So you you basically you take a a either a leaf punch, preferably a piece of root because some plants hide it in the roots and it's not in the canopy. And you boil that for a little bit and then run it through PCR.

And RT PCR will give you an answer, and we've got a system that does it like in 50 minutes, you get out an answer as to whether it's positive or not. If it's positive, in most cases, they they just call the plants. There are a few, like, mother plants where people in the cannabis industry, they're not using seeds as much for THC plants that are grown indoor because, there's too much variability right now in in a lot of the seeds. Seeds are siblings, so they're not all the same. Clones are identical to the mother.

So what happens on an expensive indoor cannabis grow is they clone the mother plants. And that cloning process is cutting into the plant quite a bit and creating a lot of injury. And that's where a lot of the hoplatin virology is getting transmitted through. It's getting transmitted through scissors. So that cloning process can spread it very, very quickly in a nursery.

So there are some studies right now that we're working on that show there are genetics out there that seem to be more tolerant of this. So you want to be careful. If you put a program in place where you just chop everything down that's positive, you may be chopping down some of the plants that you need to breed to get out of this. There are some plants that keep it in the roots, and it doesn't go to the canopy. And those are probably the ones you want to start breeding with to build up a set of genetics that are more tolerant to this.

What what strains would those be? We've seen that in Jamaican lion, which is one that, we've done a lot of publishing on. It keeps it in the roots, and doesn't spread to the canopy. Zamere punja has a few strains as well. They're mentioned in his recent publication that had maybe only 5% yield loss and very rarely had it in the canopy as well.

I don't know the actual names of his strains. They're in his paper, but I don't remember them offhand. So And how how how big of a problem has this been for growers so far? Well, it's, I think it's more of an issue on the indoor grows where there's a lot of cloning going on. And so we've heard numbers of 40% yield loss.

We've heard, you probably a $1,000,000,000 of loss in California alone. People are getting better at this. Those reports are a little bit old, and they're not of the places that are doing a lot of the cloning have to have better sterility and more bleach involved in the process. So once people have put molecular testing in place, they can get the infection rates down to almost 0. They just have to have an aggressive screening program every time they bring new genetics in.

There's a little bit of work going on with tracking this in seeds and in pollen. It does move in pollen. It's not as effective in the seeds. Some lines have a higher percentage of seeds getting it than others. So there's still a lot of work to be done on which genetics move more of this than others.

I think we're going to find in time that some of them are more The hot field went through this already, and and they don't test anymore because they've now got the lines that they need to to basically and they know which lines are more tolerant of it, and they they tend to grow those. I think that I see. So so people may sort of breed breed the problem away by creating plants that are more resistant? I think that's yeah. And and that's an important point that if if like if you have these plants that are positive in their roots but they never move to the canopy and you kill those 2, you might be killing the plants that we actually need to keep and breed. So we've always recommend people test both leaf and root to know what kind of plant you have. And you got to do it in veg and in flower because in flowering the plant changes and you end up getting more thyroid flow in some of the plants. But if you don't have it in the flowers and it's in the roots, that may not be one mother plant you want to kill. That might be one you want to put in your breeding program because it seems to be keeping the thyroid contained at least. Alright, Kevin.

I've taken, probably enough of your time here. Is there anything that you want to reiterate or any final thoughts you want to leave people with about anything that we we talked about today? No. I want to thank you actually for the time on this. I I honestly think this is the, people get a better sense of what's going on in the scientific field from long form casts like yours, where you can dissect these things because it's very difficult, I think, for the average person now to comb through the peer reviewed literature and know, okay, which papers are conflicted, which ones aren't, what does this all mean in layman's terms.

So you guys are serving a very important role, I think, in in distilling a lot of this, arcane language into what everyone else can understand. So hats off to you for that. If anyone needs to wants to follow our work, you can find us. I'm at Medicinal Genomics. You can find me on Twitter, and, I have a substack that, presents a lot more of this controversial work.

And, unfortunately, I made the worst possible name for my sub stack because no one can pronounce it, but, it's nipedalactone. If you ever get lost, it's the compound that's, the active ingredient in catnip. That'll lead you there. Alright. Kevin McKernan Kevin McKernan, thank you for your time.

Thanks, Nate. Appreciate it.